**42. Arf6 regulates endometriotic epithelial-mesenchymal transition and mitochondrial distribution**

[Article in Chinese]

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**Abstract**

**Objective:** To investigate the role of adenosine diphosphate ribosylation factor 6 (Arf6) in the pathogenesis of endometriosis. **Methods:** Endometrial tissues were sampled from women who were hospitalized in the Affiliated Hospital of Medical School of Ningbo University and Ningbo Women and Children's Hospital from November 2020 to May 2021 with endometriosis (*n*=44, endometriosis group) and without endometriosis (*n*=17, control group). The expression of Arf6 protein in the endometrial tissues was detected by western blot. Endometrial epithelial cells from both groups were primary cultured and the distribution of intracellular mitochondria was detected by immunofluorescence. The expression of Arf6 protein was down-regulated by small interference RNA (siRNA), the distribution of mitochondria in cells with decreased Arf6 protein expression was observed, and the expression of mitochondria-related proteins development and differentiation enhancing factor 1 (DDEF1, also called AMAP1), reactive oxygen species 1 (ROS1) and epithelial- mesenchymal transition (EMT)-related proteins E-cadherin, vimentin were detected. Transwell assay was used to detect the changes in the migration ability of the cells. **Results:** Compared with the control group, ectopic endometrial tissue of endometriosis group showed high expression of Arf6 protein (0.174±0.019 vs 0.423±0.033; *t*=29.630, *P*<0.01); and in ectopic endometrial epithelial cells, mitochondria were distributed near the edge of the cell membrane. While Arf6 expression was down- regulated by siRNA, the distribution of mitochondria in ectopic cells returned to natural, close to the control level. In addition, the expression levels of AMAP1 and ROS1 in ectopic cells after Arf6 protein knockdown were significantly decreased. Transwell assay results indicated that knockdown of Arf6 could reduce the migration ability of ectopic epithelial cells [migration cell count: (34.3±7.5) cells]; and immunofluorescence verified low expression of E-cadherin but high expression of vimentin in ectopic epithelial cells, whereas knockdown of Arf6 protein E- cadherin expression increased but vimentin expression decreased. **Conclusions:** High expression of Arf6 protein in ectopic endometrial epithelial cells leads to the distribution of mitochondria tending to membrane marginalization, while inducing EMT, which are involved in the mechanism of endoheterosis pathogenesis.